Correspondence

Archives of Disease in Childhood, 1975, **50**, 250.

Ethylene chlorohydrin intoxication

Sir,

Miller, Dobbs, and Jacobs (*Archives*, 1970, **45**, 589) reported the fatal outcome of ingestion of 1–2 ml of ethylene chlorohydrin by a 23-month-old child. The common name of this chemical may obscure the fact that it is the 2-chloro derivative of ethanol. Both Peterson, Peterson, and Hardinge (1968) and I (1965) have shown that oral dosage with ethanol reduces the toxicity of 2-chloroethanol in rats. Repeated high doses of ethanol would seem to be part of the rational therapy of this intoxication. Forced diuresis might also help.

Chloroacetic acid is not highly toxic but chloroacetaldehyde produced intracellularly would be very reactive and is probably the actual toxic agent (Johnson, 1967; see also Merck Index, 1968).

Inclusion of ethanol in chemical compositions containing ethylene chlorohydrin would substantially lower their toxicity.

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Possible aetiology of hyperglycaemia in hyperosmolar dehydration

Sir,

In their report of 6 cases of hyperosmolar dehydration with accompanying hyperglycaemia, Heggarty, Trindade, and Bryan (*Archives*, 1973, **48**, 740) do not give details of how the infants were being fed before and during their illnesses. An infant recently seen in this hospital is of interest.

A 10-week-old boy was admitted collapsed, with a rapid respiratory rate, after a 24-hour illness of mild diarrhoea and vomiting. Plasma electrolytes were: sodium 175 mEq/l; potassium 4.4 mEq/l; urea 205 mg/100 ml. He was treated with intravenous fluids and made an uneventful recovery.

Until 12 hours before admission he had been fed on full-cream dried milk. His mother levelled the scoops of powder by pressing the powder down with a spoon, after dragging the scoop up the side of the packet. She regularly added 1 teaspoon of glucose to a 5 oz (142 ml) feed. The osmolality of the resulting milk was 990 mOsm/kg and it contained Na 51 mEq/l. When he became ill his mother gave him 'glucose water' made by adding one teaspoonful of glucose powder to one ounce (30 ml) of boiled water. The osmolality of the resulting solution was 1065 mOsm/kg. (The osmolalities of 1/5 normal saline/4.3% dextrose and 20% dextrose are 303 mOsm/kg and 1312 mOsm/kg, respectively.)

The problem of solute handling by the infant kidney is well recognized. It seems that the solute load presented by this baby's feeds caused it to be in a continual state of relative water deprivation. Gastroenteritis, which might not have taxed a better hydrated baby so severely, caused him to become rapidly hypernatraemic. The glucose in the regular milk would have contributed to the hyperosmolality: that given during his illness can hardly have relieved the situation. Though the blood glucose and pH were not measured, it can be seen how hyperglycaemia could have occurred because of persistent administration of glucose in the presence of a probable metabolic acidosis, which, as suggested by Heggarty *et al.*, would tend to inhibit its utilization. Hypernatraemia and uraemia, predisposed to in this baby by the pre-existing renal water requirements for solute excretion, may have further contributed to the hyperglycaemia by their possible promotion of glucose intolerance and increased gluconeogenesis, as described by Nitzan (1974).

A further point is that this mother had given the glucose water because she knew that ill babies need more dilute feeds. Surely our message should be that what babies need in these situations is boiled water.

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